

Journal Resources

Morphologic changes in thyroid glands of puppies fed a high-iodine commercial diet

Iodine excess causes alterations in thyroid activity, blocking both its characteristic functions and cell proliferation. Depending on the dose of iodine and on the previous conditions of the gland, iodine excess can have a goitrogenic effect and induce the blockade of hormone biosynthesis and secretion-provoking hypothyroidism.

Three groups of puppies younger than 3 months old were fed different diets: 1) a home-prepared diet (control group), 2) a commercial diet (containing 5.6 mg potassium iodide/kg dry food), and 3) a home-prepared diet supplemented with 5.6 mg potassium iodide/kg dry food. Thyroid volume was evaluated by sonography and by weight, histopathology, and morphometry, and thyroid hormones were measured (thyroid stimulating hormone [TSH] and thyroxine T4). Volume, weight, and diameter of the thyroid follicles were increased ($p < 0.05$) in the 2 iodine-supplemented groups as compared with the control group. TSH was increased ($p < 0.05$) and T4 was lower ($p < 0.05$) in groups with a high amount of iodine in diets than in the control group.

Conclusions and Clinical Relevance Increased dietary iodine alters thyroid morphology and function in puppies younger than 3 months old. Feeding excessive amounts of iodine in foods and dietary supplements (kelp, seaweed) could reduce thyroid function and perhaps contribute to the present day prevalence of hypothyroidism in young adult dogs.

Reference: Castillo, Rodriguez, Lalia, Pisarev. *Int J Appl Vet Med* 1(1): 1-8, 2003.

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Linking iodine with autoimmune thyroiditis

Circumstantial evidence has linked iodine with the rising incidence of autoimmune thyroiditis. T-cells from humans with chronic lymphocytic thyroiditis proliferate in the presence of iodinated but not non-iodinated human thyroglobulin. Moreover, the proliferative response is restored when the thyroglobulin is iodinated artificially *in vitro*. The presence of iodine induces a number of stereochemical changes in the conformation of the molecule, resulting in the loss of some antigenic determinants and the appearance of others. One prominent determinant was associated with the iodine-containing amino acid thyroxine. Both the number and position of the iodine substituents determine the precise specificity of this epitope.

A new model for the study of the role of iodine in inducing thyroid autoimmunity has become available in the form of the non-obese diabetic (NOD) -H2 mouse. This animal develops autoimmune thyroiditis spontaneously but in relatively low prevalence. However, if iodine is added to the drinking water, the prevalence and severity of the thyroid lesions increase markedly. The immune response is specific for thyroglobulin, both in terms of the antibody response and T-cell proliferation. In fact, the appearance of lesions can be predicted by the presence of thyroglobulin-specific IgG2b antibody. The disease, moreover, can be transferred adoptively, using spleen cells from iodine-fed donors treated *in vitro* with iodinated thyroglobulin. Based on T-cell proliferation, it appears that the NOD-H2 strain of mice has innately a greater response to murine thyroglobulin than do other mouse strains, and that the proliferation is increased even more by feeding iodine.

Conclusions and Clinical Relevance The presence of iodine increases the autoantigenic potency of thyroglobulin, a major pathogenic antigen in the induction of autoimmune thyroiditis. The non-obese diabetic mouse serves as an animal model for investigating the mechanisms whereby an environmental agent can trigger a pathogenic autoimmune response in a susceptible host.

Reference: Rose, Rasooly, Saboori, Burek. *Environ Health Perspect* 107(suppl 5):749-752, 1999.

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